

**EFFECT OF BASELINE ST SEGMENT ELEVATION ON STANDARD AND HEART RATE ADJUSTED ST SEGMENT DEPRESSION CRITERIA**

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Whether the ST segment shift used to evaluate the presence and severity of coronary disease (CAD) should include the additional deviation due to decreasing amounts of baseline ST segment elevation was examined in 100 clinically normal subjects and in 124 pts with CAD. Exercise ST segment depression was calculated in 2 ways: as the difference between exercise and resting ST depression, excluding any resting ST elevation (STdep), and as the total ST segment difference including any resting ST elevation (STdiff). These values were also used for calculation of the maximal ST/HR slope and delta ST/HR index. At partition values with matched specificity of 95% in clinically normal subjects, 150  $\mu$ V of STdep was significantly more sensitive for identification of pts with CAD than was 220  $\mu$ V of STdiff [61% (76/124) vs 50% (62/124),  $p < 0.005$ ]. Comparison of receiver operating characteristic curves confirmed the superior test performance of STdep for the identification of CAD (area under the curve 0.920 vs 0.869,  $p = 0.0019$ ). In contrast, detection of 3-vessel CAD by standard ST segment criteria was not affected by definition of ST segment excursion. Substitution of STdiff for STdep did not change the performance of the ST/HR slope or the delta ST/HR index for either the detection of CAD or for the identification of 3-vessel CAD. We conclude that incorporation of resting ST segment elevation into the measurement of exercise-induced ST segment depression decreases the sensitivity of standard ECG criteria for the detection of CAD and does not improve the performance of heart rate adjusted ST segment criteria for the assessment of either the presence or severity of CAD.

**CLINICAL SIGNIFICANCE OF PROLONGED ST SEGMENT DEPRESSION AFTER EXERCISE TESTING IN CHRONIC STABLE ANGINA**

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To assess the significance of prolonged ST segment depression after exercise testing, 75 patients (pts) (59 men, 16 women) with chronic stable angina, positive exercise tests ( $\geq 0.1$  mV ST segment depression) and documented coronary artery disease were studied. All pts were exercised off antianginal therapy, with the modified Bruce protocol. Results of tests were analyzed without previous knowledge of the patients' clinical and angiographic characteristics. Exercise tests were positive in all pts. After exercise (recovery), the ST segment returned to its baseline value  $\pm 0.02$  mV within 2 minutes in 26 pts (short recovery) and after 5 minutes in 24 (long recovery). Duration of recovery was intermediate in the remaining pts. Age, sex, number of diseased vessels, ventricular function at rest and blood pressure during recovery were similar in both pts with short and long recovery. Rate-pressure product (RPP = bpm  $\times$  mmHg  $\times 10^{-2}$ ) at peak exercise, exercise time and time to 0.1 mV of ST depression were not significantly different in pts with short and long recovery (22.7 $\pm$ 7 vs 20.4 $\pm$ 8; 9.5 $\pm$ 5 vs 9.0 $\pm$ 5 min and 8.3 $\pm$ 4 vs 7.4 $\pm$ 5 min, respectively). However, pts with long recovery had both significantly lower RPP at 0.1 mV of ST depression (21.6 $\pm$ 7 vs 17.7 $\pm$ 5;  $p < 0.05$ ) and coronary stenoses of greater severity than pts with short recovery (78 $\pm$ 16% vs 88 $\pm$ 17% diameter reduction;  $p < 0.05$ ). Thus, recovery from exercise-induced myocardial ischemia is prolonged in pts with coronary stenoses of greater severity and lower ischemic threshold. Severity of coronary stenoses rather than extension of coronary artery disease is of importance for the development of prolonged ischemic ST segment depression after exercise in pts with chronic stable angina.

**SILENT MYOCARDIAL ISCHEMIA: A FUNCTION OF WORKLOAD?**

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In order to evaluate the relationship between silent ischemia (SI) and exercise intensity, 13 patients (PTS) were exercised with symptom-limited Bruce Protocol (BP) and later underwent 20 minutes of steady-state submaximal exercise (SM) at 70% of the heart rate (HR) achieved on the BP. Ischemia (I) was determined by angina pectoris (AP), S-T depression (ST), or a reversible thallium (TL) defect. Oxygen uptake (VO<sub>2</sub>; ml/kg/min) and pressure-rate product (PRP) were measured.

	ST(n)	AP(n)	TL(n)	ONSET OF ST DEPRESSION		
				HR	PRP $\times 10^3$	VO <sub>2</sub>
BP	13	9	13	117	19.4	16.2
LL	10	3*	10	98*	13.7*	11.6*

\* $p < 0.05$

Using the Bruce Protocol, all PTS had S-T depression and positive TL and 70% accompanied by angina. Using low-level exercise, those with S-T depression had positive TL, but only 23% had angina ( $p < 0.01$ ). Of those PTS with silent ischemia, the S-T depression occurred at significantly lower HR, PRP and VO<sub>2</sub>.

**CONCLUSION:** In patients with angina pectoris demonstrated by Bruce Protocol, myocardial ischemia can occur during submaximal exercise at a lower exercise intensity and often not accompanied with anginal symptoms. Therefore, patients with angina pectoris are at risk for silent myocardial ischemia at workloads in which angina is not manifest.

**POST EXERCISE OXYGEN UPTAKE KINETICS AND EXERCISE CAPACITY IN CARDIAC PATIENTS**

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To evaluate oxygen uptake ( $\dot{V}O_2$ ) kinetics after exercise (Ex), 31 normal controls and 68 Pts with congestive heart failure (CHF: 26 in New York Heart Association Functional Class I, 34 in Class II and 8 in Class III) underwent incremental cycle ergometer Ex testing with breath by breath respiratory gas exchange analysis.

For the initial several minutes after cessation of Ex,  $\dot{V}O_2$ -time relations showed an exponential decline. We fitted the  $\dot{V}O_2$ -time relation of the first 3 minutes after peak  $\dot{V}O_2$  to an exponential curve, to obtain the time constant ( $T[\dot{V}O_2]$ ) of the  $\dot{V}O_2$  decline. The correlation coefficient was above 0.96 in 28 controls and in 58 CHF Pts (mean of all 99 cases = 0.98 $\pm$ 0.01), indicating reasonable curve fitting.  $T[\dot{V}O_2]$  (msec) was 116 $\pm$ 17 for controls, 130 $\pm$ 24\* in Class I, 143 $\pm$ 26\* in Class II and 183 $\pm$ 54\* in Class III, respectively (\* $p < 0.05$  between each class), suggesting that oxygen debt is prolonged with the severity of CHF.  $\dot{V}O_2$  change per work rate, ventilatory anaerobic threshold, and peak  $\dot{V}O_2$ , which are related to cardiac function during Ex, were also correlated with  $T[\dot{V}O_2]$  ( $r = -0.75$ ,  $-0.52$  and  $-0.58$ , respectively). Thus,  $T[\dot{V}O_2]$  is a useful index in evaluating Ex capacity, which reflects cardiac function during and after Ex.